PRINCETON UNIVERSITY
PRINCETON NEW JERSEY

Department of Biology

July 7, 1943

Dear Sonneborn:

I am sorry not to have answered your letter earlier. Do not think I did not consider very carefully the objections you put forward concerning the paper on virus-resistant bacteria.

I understand that you do not object either to the experiments, or to the mathematicl theory, which, as you correctly remarked, only shows that resistant are grouped in families, and in particular in such a way as can be predicted on the assumption that every time a bacterium is resistant to virus, all of its offsprings will also be resistant. You feel, if I understand correctly, that the possibility exists, that occasionaly certain bacteria find themselves in such a physiological status that an attack by virus is followed by resistance (inheritable, at least in asexual reproduction, which is the only existant in bacteria). Moreover, this particular physiological status should appear in family groups. Let me state that, if this physiological status were always inherited, so that all offspring of a cell which has been once "potentially resistant" are also potentially resistant, I would be ready to consider it identical with our assumption. It would still be a case of stable inheritable change, and it is immaterial whether the physiological property of not having the sensitive receptors for virus only appears after the action of the virus. This is said in our discussion, with the reasons for which we believe that the physiological change takes place before and not after the action of the virus. The other possibility that I see, which would follow from your suggestion, is that the physiological status which produces resistance is not inheritable, but only occasionaly present in certain families (I should say clones), and the stable, inheritable change is produced by the attack of the virus. Besides the fact that this possibility is a little unpleasant to conceive in the case of a change which remains stable over innumerable generations, I think it would be quite peculiar that this condition should be present in such a way as to give the same distribution of resistant bacteria as predicted on the basis of the theory, according to which, I repeat, all offspring of a bacterium resistant to virus are also resistant. The vanishing away of the clonal, temporary resistance (or predisposition to it)

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should show up, I think, as a definite change in the distribution of resistant bacteria. I am going to discuss this point in correspondence with Delbruck, and hope to be able to find a more convincing, quantitative answer. I do not have with me the abstract of your paper, which remained in Bloomington. I would like to know where it is published. If you are not too busy, and want to waist more time with me, I would like very much to hear more objections, particularly, some other of the interpretations which you consider possible, besides the one that I took the liberty of attributing to you in the preceding part of this messy letter. I am scared at the idea that I am going to read it over in a moment, and probably find it incomprehensible.

I got word from Zirkle that they cannot wait any longer for my clearance to arrive from Washington, therefore I am going to be back in Bloomington in September. I got the respirometric technique well in hand, and hope to get some more insight in phage growth some day.

After an awful heat spell, we have started living again. Princeton is almost as boring as Bloomington, but New York is within easy reach.

Best regards,

Luria

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